

## Fluid dynamics approach to airway obstruction

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### ABSTRACT

**Background:** Fluid dynamics theory, which is a fundamental underlying concept applied to fluid management, has not been introduced to analyze the human respiratory system. We hypothesized that one of the potential mechanisms that promotes airflow limitation in patients with airway obstructive disease would be elucidated by using fluid dynamics theory.

**Methods:** We calculated the values of pressure loss and static pressure change under virtual tracheal stenotic conditions using the fluid dynamics approach.

**Results:** Under normal conditions, the absolute values of pressure loss and static pressure change are very low. However, once airway stenosis occurs, it is confirmed that they would be dramatically elevated.

**Conclusions:** The fluid dynamics approach to airway obstruction is very constructive. The treatment strategy for airway obstruction and the reasons for airflow limitation are well explained by using this approach.

### Introduction

Although fluid dynamics theory is a fundamental underlying concept applied to fluid management, in the field of medicine we have failed to apply this theory to the respiratory system [1]. On the other hand, the circulatory system has been well described by this theory [2]. One of the reasons for this may be due to the differences between the examination procedures used in respiratory versus cardiovascular medicine. The “dynamic” ultrasound examination has already been developed for cardiovascular medicine, whereas for respiratory medicine the “static” X-ray and computed tomography (CT) scan are still primarily used for anatomical assessment and pulmonary function tests are used for physiological assessment of the respiratory system. Now is an appropriate time to further the understanding of the respiratory system by applying fluid dynamics theory.

### Hypothesis

One of the potential mechanisms that promote airway collapse in patients with airway obstructive disease is negative intra-airway static pressure change during respiration. We hypothesized that negative intra-airway static pressure change due to high-speed flow would be elucidated by applying fluid dynamics theory.

### Fluid dynamics equations

The continuity equation and the Bernoulli equation for steady frictionless incompressible flow imply the following:

$$Q = Au \quad (1)$$

$$p + \frac{1}{2}\rho u^2 + \rho gz = \text{const.} \quad (2)$$

where:

$Q$  is flow rate [ $\text{m}^3/\text{s}$ ],

$A$  is cross-sectional area [ $\text{m}^2$ ],

$u$  is the fluid flow velocity at a point on a streamline [ $\text{m/s}$ ],

$p$  is the static pressure at the chosen point [ $\text{Pa}$ ] ( $= [\text{N}/\text{m}^2] = [\text{kg}/\text{m}\cdot\text{s}^2]$ ),

$\rho$  is the density of the fluid at all points in the fluid [ $\text{kg}/\text{m}^3$ ],

$g$  is the acceleration due to gravity [ $\text{m}/\text{s}^2$ ], and

$z$  is the elevation of the point above a reference plane [ $\text{m}$ ].

When removing the effect of gravity, the following equation is gained:

$$p + \frac{1}{2}\rho u^2 = \text{const.} \quad (3)$$

where “ $\frac{1}{2}\rho u^2$ ” is known as “dynamic pressure”.

Formula (3) represents the law of conservation of fluid energy in units of pressure. In considering pressure loss “ $\Delta p$ ” due to flow resistance, formula (3) is rewritten as follows:

$$p_1 + \frac{1}{2}\rho u_1^2 - \Delta p = p_2 + \frac{1}{2}\rho u_2^2 \quad (4)$$

where:

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$p$  is the static pressure at the chosen point [kg/m<sup>2</sup>·s<sup>2</sup>],  
 $\rho$  is the density of the fluid [kg/m<sup>3</sup>],  
 $u$  is the fluid flow velocity at a point on a streamline [m/s],  
 subscripts 1 and 2 refer to two positions along a streamline, and  
 $\Delta p$  is the pressure loss of fluid from positions 1 to 2 due to flow resistance.

The Reynolds number “ $Re$ ” is defined as:

$$Re = \frac{\rho u L}{\mu} = \frac{u L}{\nu} \tag{5}$$

where:

$\rho$  is the density of the fluid [kg/m<sup>3</sup>],  
 $u$  is the velocity of the fluid [m/s],  
 $L$  is a characteristic linear dimension [m],  
 $\mu$  is the dynamic viscosity of the fluid [kg/m·s], and  
 $\nu$  is the kinematic viscosity of the fluid [m<sup>2</sup>/s].

Laminar flow occurs at low Reynolds numbers ( $Re < 2300$ ), where viscous forces are dominant, and is characterized by smooth, constant fluid motion. Turbulent flow occurs at high Reynolds numbers ( $Re > 2900$ ) and is dominated by inertial forces, which tend to produce chaotic eddies, vortices, and other flow instabilities.

In a cylindrical pipe of uniform diameter, flowing full, the pressure loss “ $\Delta p$ ” due to viscous effects can be characterized by the Darcy–Weisbach equation as follows:

$$\Delta p = \lambda \cdot \frac{l}{d} \cdot \frac{1}{2} \rho \bar{u}^2 \tag{6}$$

where:

$\lambda$  is the flow coefficient,  
 $l$  is the length of the pipe [m],  
 $d$  is the diameter of the pipe [m],  
 $\rho$  is the density of the fluid [kg/m<sup>3</sup>], and  
 $\bar{u}$  is the mean fluid flow velocity [m/s].

By formula (1), the mean fluid flow velocity “ $\bar{u}$ ” in the cylindrical pipe is calculated as follows:

$$\bar{u} = \frac{Q}{A} = \frac{Q}{\pi \left(\frac{d}{2}\right)^2} = \frac{4Q}{\pi d^2} \tag{7}$$

Then, formula (6) is rewritten as follows:

$$\Delta p = \frac{8\lambda\rho}{\pi^2 d^5} \cdot Q^2 \tag{8}$$

Adding an explanation regarding flow coefficient “ $\lambda$ ”, for laminar flows ( $Re < 2300$ ), it is a consequence of Poiseuille’s law as follows:

$$\lambda = \frac{64}{Re} \tag{9}$$

In this article, for critical or turbulent flows ( $Re \geq 2300$ ), “ $\lambda$ ” is calculated by using the goal seek function in Excel® from the Colebrook–White relation as follows:

$$\frac{1}{\sqrt{\lambda}} = -2 \log_{10} \left( \frac{\varepsilon/d}{3.71} + \frac{2.51}{Re\sqrt{\lambda}} \right) \tag{10}$$

where:

$\lambda$  is the flow coefficient,  
 $\varepsilon$  is the roughness height of the pipe [m],  
 $d$  is the diameter of the pipe [m], and  
 $Re$  is the Reynolds number.

In a cylindrical pipe, the diameter of the pipe is used as a characteristic linear dimension for calculation of the Reynolds number. Then, for laminar flows, the following equation is gained by applying formulas (5), (7)–(9):

$$\Delta p = \frac{128\mu l}{\pi d^4} \cdot Q \tag{11}$$

Formula (11) means that the pressure loss due to flow resistance is proportional to the flow rate for laminar flow. However, for turbulent flow, the pressure loss is not proportional to the flow rate and is larger than that for laminar flow.

It is known that the equations for fluids may be adopted for gases when the airflow is relatively slow. If the airflow is fast, then the correction due to the volume compressibility must be taken into consideration. The Mach number is a dimensionless quantity representing the ratio of flow velocity past a boundary to the local speed of sound.

$$M = \frac{u}{c} \tag{12}$$

where:

$M$  is the Mach number,  
 $u$  is the local flow velocity with respect to the boundaries [m/s], and  
 $c$  is the speed of sound in the medium [m/s].

Whereas all flows are compressible, flows are usually treated as being incompressible when the Mach number is less than 0.3. Therefore, in this article, the previously mentioned equations for fluids are adopted for humid air when the value of flow velocity is less than 100 m/s (because the value of the speed of sound in humid air at 36 °C is approximately 350 m/s).

### Calculations

What would be the values of flow velocity, dynamic pressure, pressure loss, static pressure change, and the Reynolds number in a clinical situation of airway obstruction? As an example, a case of a virtual one-year-old infant, whose body weight is 10 kg, body height is 75 cm, crown-rump length is 50 cm, tracheal internal diameter is 6.4 mm, forced peak inspiratory flow rate is 30 L/min, and peak inspiratory flow rate at rest is 5 L/min, with tracheal stenosis is shown as a stenotic tracheal model (Fig. 1).

As preconditions,

The value of the humid air density at 36 °C is 1.117 kg/m<sup>3</sup>.

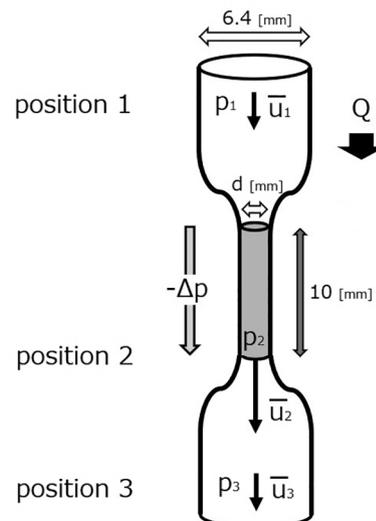
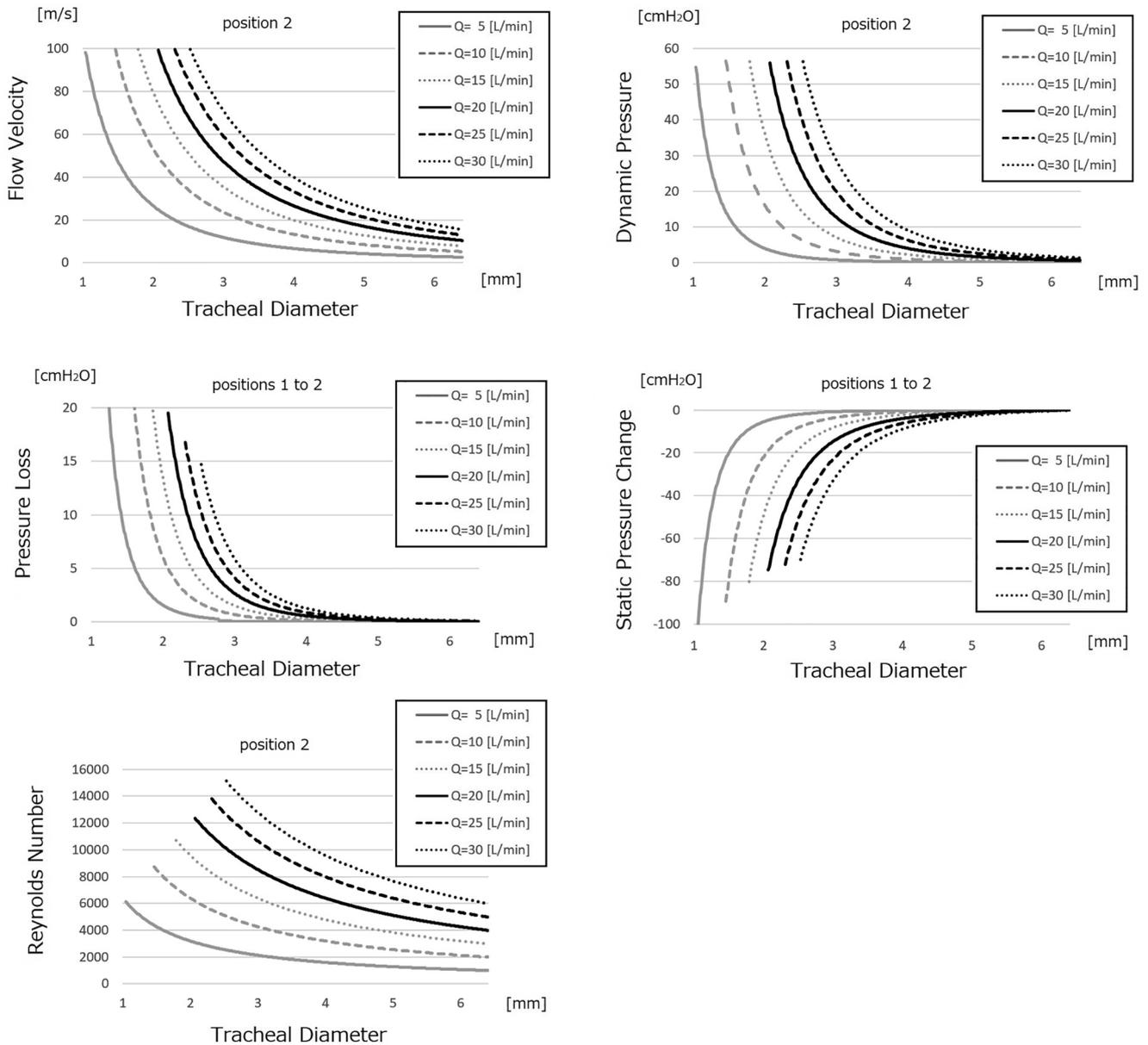


Fig. 1. Stenotic tracheal model.



**Fig. 2.** Calculation results. Flow velocity at position 2, dynamic pressure at position 2, pressure loss from positions 1 to 2 due to flow resistance, static pressure change from positions 1 to 2, and the Reynolds number at position 2 are calculated using formulas (5), (7)–(10), and (14). Laminar flow occurs at low Reynolds numbers ( $Re < 2300$ ) and turbulent flow occurs at high Reynolds numbers ( $Re > 2900$ ).

The value of the humid air kinematic viscosity at 36 °C is  $1.662 \times 10^{-5} \text{ m}^2/\text{s}$ .

A short segment of the trachea with changeable diameter is considered and the value of its length is determined as 10 mm ( $=1.000 \times 10^{-2} \text{ m}$ ). The segmental trachea shape is considered as a cylindrical pipe.

During calculations, the value of segmental tracheal diameter is variable in the range of 1 to 6.4 mm ( $=1.000 \times 10^{-3}$  to  $6.400 \times 10^{-3} \text{ m}$ ) by intervals of 0.01 mm.

The value of the roughness height of the segmental trachea is 0.1 mm ( $=1.000 \times 10^{-4} \text{ m}$ ) despite the tracheal diameter.

The value of flow rate is also changed in the range of 5 to 30 L/min ( $=8.333 \times 10^{-5}$  to  $5.000 \times 10^{-4} \text{ m}^3/\text{s}$ ) by intervals of 5 L/min during calculations.

The calculated result is regarded as being valid if the value of flow velocity is less than 100 m/s.

For the virtual trachea of the hypothetical pediatric patient, the

values of flow velocity, dynamic pressure, pressure loss, and the Reynolds number are calculated using formulas (5), (7)–(10).

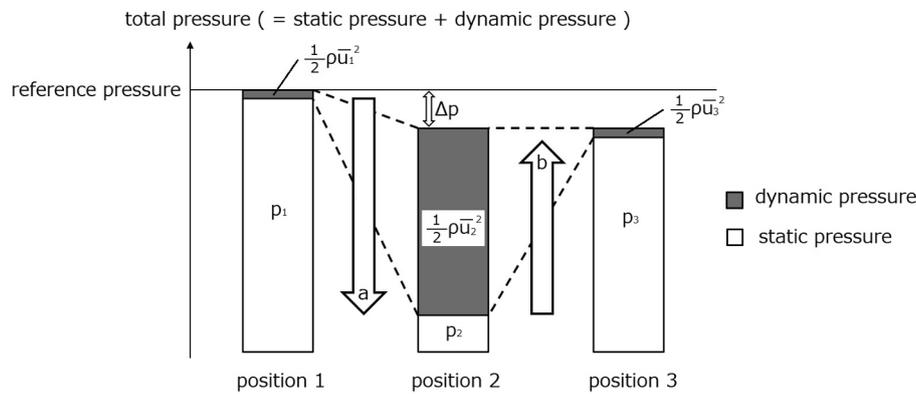
The static pressure change “ $p_c$ ” from positions 1 to 2 is defined as follows:

$$p_c = p_2 - p_1 \tag{13}$$

By formula (4), “ $p_c$ ” is rewritten as follows:

$$p_c = \frac{1}{2}\rho u_1^{-2} - \frac{1}{2}\rho u_2^{-2} - \Delta p \tag{14}$$

The results for flow velocity at position 2, dynamic pressure at position 2, pressure loss from positions 1 to 2 due to flow resistance, static pressure change from positions 1 to 2, and the Reynolds number at position 2 are summarized as graphs (Fig. 2). The schematic chart of total pressure, which is the sum of static pressure and dynamic pressure, at positions 1, 2, and 3 is also shown in Fig. 3.



**Fig. 3.** Total pressure at positions 1, 2, and 3.  $\Delta p$  is the pressure loss from positions 1 to 2 due to flow resistance. Arrow “a” represents the static pressure change from positions 1 to 2 and arrow “b” represents the static pressure change from positions 2 to 3.

**Discussion**

As gas exchange in the human lung occurs through mass transport by fluids, fluid dynamics is necessary for understanding respiratory physiology [1]. However, the respiratory system has been modeled according to an electric circuit analogy [3]. The fluid dynamics approach has been lacking in the field of respiratory medicine [1], whereas the approach is used for the circulatory system [2]. The major problem with the electric circuit model for the respiratory system is that both the dynamic motions of the airway structure and the changes in airway resistance are not considered. For laminar flow in a cylindrical pipe of uniform diameter, by formula (11), the pressure loss is proportional to the flow rate and the proportion coefficient can be thought of as the resistance of the pipe, as follows:

$$R = \frac{128\mu l}{\pi d^4} \tag{15}$$

where:

- $R$  is the resistance of the pipe [kg/m<sup>4</sup>·s],
- $\mu$  is the dynamic viscosity of the fluid [kg/m·s],
- $l$  is the length of the pipe [m], and
- $d$  is the diameter of the pipe [m].

However, for turbulent flow, the resistance of the pipe is not fixed while the flow rate is changing. In this article, the values of flow velocity, dynamic pressure, pressure loss, static pressure change, and the Reynolds number for the virtual trachea of the imaginary pediatric patient were calculated. In most calculated cases, the Reynolds numbers were greater than 2900. Turbulent airflow mainly occurs intratracheally, particularly when the airway is stenotic. For these reasons, the electric circuit model of the respiratory system does not facilitate analysis of airway obstruction.

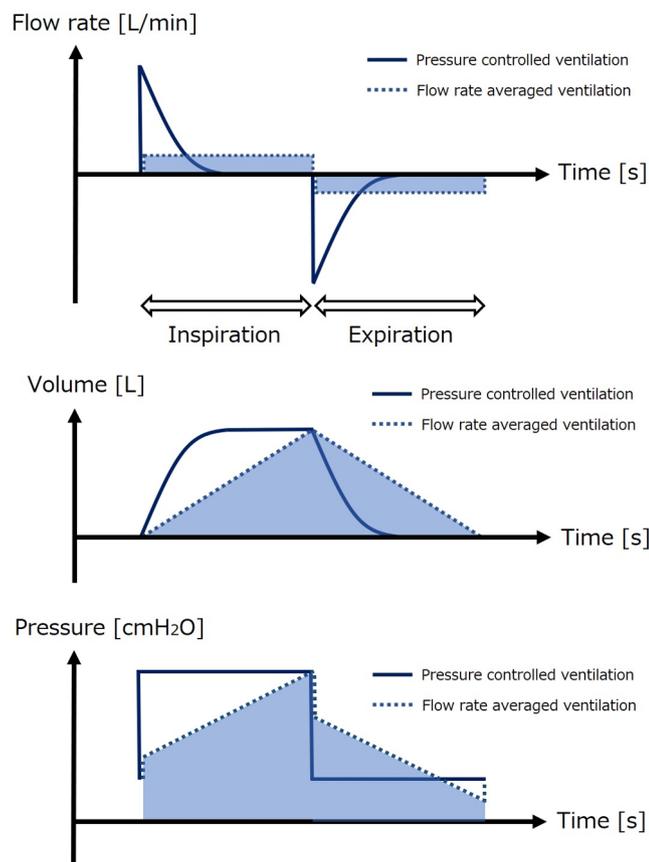
For laminar flow, the theoretical increase in flow resistance as tube diameter decreases is obvious, as it is inversely proportional to tube diameter to the fourth power in the denominator of equation (15). For turbulent flow, however, it is known that flow resistance becomes even higher. It was reported that the measured pressure loss with a flow rate of 30 L/min through the endotracheal tubular parts with an internal diameter (ID) of 6.0 mm and length of 263.8 mm was 3.06 cmH<sub>2</sub>O, and for an ID of 8.0 mm and length of 306.5 mm it was 1.02 cmH<sub>2</sub>O [4]. When correcting for tube length with 8.0 mm ID to be equivalent to tube length with 6.0 mm ID, the pressure loss of the tube with 8.0 mm ID was calculated as 0.88 cmH<sub>2</sub>O. The increase in flow resistance is due to the tube diameter being raised to the power of 4.34 in this turbulent flow situation.

Normal measurements for the trachea of children related to crown-rump length have been reported [5]. The tracheal internal perimeter of

a one-year-old infant with a crown-rump length of 50 cm is estimated to be approximately 20 mm. Assuming the trachea is circular, the tracheal diameter is calculated as approximately 6.4 mm. Tracheal diameter of a one-year-old infant can be also estimated by appropriate endotracheal tube size for patients of this age. For a one-year-old infant, we choose a 4.5 mm ID (6.2 mm outer diameter) uncuffed tube; there is usually a slight tube air leak. Forced inspiratory and expiratory flows in healthy young children were previously described [6]. Mean peak inspiratory flows were approximately 70 L/min in a 3-year-old group, 81 L/min in a 4-year-old group, 99 L/min in a 5-year-old group, and 115 L/min in a 6-year-old group. It was also reported that the ratio of forced peak inspiratory flow rate to forced peak expiratory flow rate in all these groups was 0.67:1. It seems difficult to measure forced inspiratory and expiratory flow in younger healthy infants. However, forced expiratory flow could be measured by forced expiratory maneuvers for infants, and it was reported that forced expiratory flow measured at 50% expired volume (FEF<sub>50</sub>) was highly correlated with body length [7]. For example, FEF<sub>50</sub> in a one-year-old infant who was 75 cm tall was estimated to be approximately 42 L/min. If the ratio of forced peak inspiratory flow rate to forced peak expiratory flow rate is also 0.67:1 in a one-year-old infant, then forced peak inspiratory flow would be calculated as approximately 28 L/min. Inspiratory and expiratory flow at rest in a one-year-old infant who is 10 kg in weight can be estimated as follows. Assuming that the normal tidal volume at rest is approximately 60 mL (6 mL/kg), inspiratory time of one respiratory cycle is 0.6 s, expiratory time of one respiratory cycle is 1.4 s, and the infant breathes at an average flow rate, inspiratory and expiratory flows are calculated as 6 L/min and 2.6 L/min, respectively. Therefore, the ranges of input flow parameters used and the obtained results of simulations can refer to real clinical values under normal and exercise airflow conditions.

Under normal conditions (not stenotic conditions), the absolute values of pressure loss and static pressure change in the tracheal lumen made by humid air are very low. However, if once tracheal stenosis occurs, they would be dramatically elevated (Fig. 2). A negative static pressure change large in absolute value may cause collapse of the airway. That is the reason for tracheal collapse during respiration in patients with tracheomalacia.

The respiratory driving pressure is theoretically divided into the pressure consumed through the airway and the pressure used for expanding the lung. (Note: the pressure consumed through the airway is “pressure loss” and not “static pressure change.”) To maintain the flow rate despite increasing pressure loss, a higher driving pressure would be necessary. In other words, if driving pressure were not changing as the airway becomes thinner, the flow rate would be limited. During respiration at rest in a healthy human, before inspiration begins, the pressure in the pleural space is -5 cmH<sub>2</sub>O, and at end-inspiration, it reaches approximately -10 cmH<sub>2</sub>O. The normal respiratory driving pressure is approximately 5 cmH<sub>2</sub>O. In our calculations, pressure loss



**Fig. 4.** Flow rate averaged ventilation. The curves of flow rate, volume, and pressure during a respiratory cycle in the conceptual ventilation mode “flow rate averaged ventilation” are shown.

becomes higher than 5 cmH<sub>2</sub>O in rapid airflow when the tracheal diameter is less than approximately 3 mm for a hypothetical pediatric patient; this may cause clinically significant problems.

By using formulas (8) and (14), developing a treatment strategy for airway obstruction can be explained as follows:

- a. Adding additional pressure to overcome pressure loss and negative static pressure change  
We have traditionally added positive airway pressure for these patients by using a ventilator, non-invasive positive pressure ventilation, and high-flow nasal cannula.
- b. Decreasing inspiratory gas density  
Using a low-density gas (e.g., Heliox®) may be useful for these patients.
- c. Decreasing airflow rate  
It is known that light sedation is effective to relieve symptoms for pediatric patients who suffer from respiratory distress due to airway stenotic disease such as croup or tracheomalacia. Although sedatives may have adverse effects on upper airway patency, the decreasing flow rate would lead to decreasing pressure loss, and prevent the airway from collapsing by the decreasing absolute value of the static pressure change. Because it is difficult for pediatric patients to breathe slowly while they have respiratory distress, light sedation is especially effective for them.
- d. Expanding the airway  
We also use airway-expanding agents for patients with airway obstruction.
- e. Bypassing the stenotic point or hardening the airway  
Endotracheal tubes are so rigid that they would not become thinner even if the intra-tube pressure drops. However, a thin and long

endotracheal tube would create pressure loss.

For a pediatric patient with tracheomalacia whose trachea is collapsing during respiration, high positive end expiratory pressure (PEEP) therapy (approximately 10 cmH<sub>2</sub>O) is introduced. However, there are some patients for whom high PEEP therapy is not effective. In these patients, negative static pressure change due to increasing dynamic pressure is so large that additional PEEP cannot overcome the negative static pressure change. Fig. 2 demonstrates that the absolute value of static pressure change shows results up to 60 cmH<sub>2</sub>O when the airway is severely stenotic.

The large negative static pressure change may play a role in the incidence of airway oedema, such as post-extubation laryngeal oedema. It may cause fluid aspiration to the oedematous area, thus increasing the oedema and tightening the airway. It is known that post-extubation laryngeal oedema often occurs some minutes after extubation [8].

It was reported that between two groups of adult patients with chronic obstructive pulmonary diseases (COPD), nearly half of them had collapsed tracheas during their respiration [9,10]. It was thought that tracheomalacia coexisted with COPD in these patients. Kitaoka excellently explained tracheal collapse in COPD patients by fluid dynamics [1]. The combination of positive pressure from over-inflated lungs in COPD compressing the tracheal wall from the outside after the beginning of expiration and the negative intratracheal static pressure change made by rapid expiratory flow make the trachea collapse. Kitaoka also confirmed this phenomenon by dynamic 3D-CT and remarked that the collapsed part was the membranous portion of the trachea. This phenomenon is due to high-speed airflow through the trachea and is never observed by the usual static CT images taken under breath-hold conditions.

The critical clinical situation is at the moment of the airway collapse due to negative static pressure change overcoming the rigidity of airway structure. Therefore, if the airway structure is not firm, as in tracheomalacia, the small negative static pressure change may cause airway collapse. Once an airway collapses, airflow would be limited, but flow velocity would not decrease due to the cross-sectional area of the stenotic airway becoming smaller. Under these conditions, airway collapse would not be reversed until the airflow stops.

Higher respiratory driving pressure, as occurs when crying, during forced respiration, and in any ventilatory high-pressure setting, may result in critical dynamic pressure generating airway collapse. For ventilated patients with severe airway obstruction who require paralysis by neuromuscular blockage, the ventilatory strategy of minimizing both inspiratory and expiratory peak flow rates may be advantageous because it results in minimizing the absolute values of pressure loss and static pressure change through the airway. We named the new conceptual ventilation mode “flow rate averaged ventilation” (Fig. 4).

Retractive breathing seen in patients with airway obstructive diseases is due to limitation of airflow. Retraction occurs when the intrathoracic pressure becomes excessively negative compared with the atmospheric pressure and is caused by limitation of airflow. When considering limitation of airflow, the pressure loss through the airway and the dynamic changes in the airway structure may play important roles. It is therefore difficult to accurately understand respiratory physiology without the fluid dynamics theory.

**Limitations**

This article has several limitations. First, the minor pressure losses in such components as expansion, contraction, bends, and others were not considered. However, it is known that the minor pressure losses are proportional to the dynamic pressure in the flow. Therefore, even if the minor pressure losses had been considered, the treatment strategy would not have changed. Second, we had no actual patient data for this article.

## Conclusions

The fluid dynamics approach to airway obstruction is very constructive. The treatment strategy for airway obstruction and the reasons for airflow limitation are well explained by using this approach.

## Role of the funding source

No external funding has been received for this work.

## Declaration of Competing Interest

There are no conflicts of interest to declare.

## Acknowledgment

We wish to thank the medical editors from the Division of Education for Clinical Research at NCCHD, Japan for editing this manuscript.

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